

The Many Ailments of Clover

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There are some 250 described species of true clovers (*Trifolium*) but only four species—red, alsike, white (including Ladino), and the crimson—are widely grown and of great importance.

The swectclovers (*Melilotus*) are not true clovers. Twenty-two species of sweetclover are recognized. Three species—white, yellow, and sour clover—are of importance in agriculture.

All clovers are subject to injury from diseases. All parts of the plant are attacked and sometimes destroyed—

the roots, crowns, stems, leaves, and inflorescences. Fungi, bacteria, and the viruses all can damage the clovers.

Some of the pathogens infect only specific organs of the plant, such as the leaves or roots. Others attack several or all parts of a plant. The pathogens differ also in parasitism. Some infect only certain species of clover. Others have a broad range of hosts and can attack nearly all clovers and many other hosts as well.

An important problem in producing clovers, the establishment and maintenance of stand, involves several factors. One is the root and crown disease complex, which includes the seedling blights, root rots, and crown rots.

Those diseases probably are the most important of all clover diseases. They occur wherever clovers are grown. They are caused by a complex of soil inhabiting fungi. The fungi may

be widely distributed or occur only locally. Some are virulent pathogens that can attack vigorous plants. Others are weak pathogens that cause damage only after the plants have been weakened by winter injury, nematodes, insects, drought, unfavorable soil conditions, or improper management. Some are primarily seedling pathogens. Others attack clover plants of any age. Some are primarily root pathogens. Others are primarily crown pathogens. Several organisms may attack a plant simultaneously, or one may follow another in sequence. Thus the difficult problem of root and crown disease is one that must receive greater attention if productive stands are to be maintained.

Crown wart of clover, caused by Urophlyctis trifolii, occurs in central Europe on red clover (Trifolium pratense), white clover (T. repens), and some others. In the United States the disease is of minor importance. It occurs mainly in the South Central States and on excessively wet soils. It is similar to the more important crown wart of alfalfa. Its characteristic symptom is the formation of irregularly shaped galls around the crown of the plant, at and just below the soil level. The galls first become noticeable in late spring and increase in size as summer advances. Infected plants wilt in hot weather. Leaves of white clover are sometimes distorted.

Sclerotinia crown and stem rot is caused by Sclerotinia trifoliorum, and is widely distributed, especially in the regions of mild winters or heavy snow cover. It has long been recognized as one of the most destructive diseases of clover in northern Europe. It occurs also in the Soviet Union and Canada. In the United States it is of considerable economic importance in the southern and central clover belts and causes extensive damage in the Pacific Northwest and in the Northeast. Rarely does it occur in the north central part of the northern clover belt. The disease spreads and develops most

rapidly during cool—55° to 65° F.—wet weather, but the fungus that causes it can grow and infect plants at temperatures ranging from below freezing to 75°.

Sclerotinia trifoliorum has a broad range of hosts, which include all important true clovers and the sweet-clovers, alfalfa (Medicago sativa), black medic (M. lupulina), birdsfoot trefoil (Lotus corniculatus), sainfoin (Onobrychis viciaefolia), and many other legumes and nonlegumes, including numerous weeds. Red clover, crimson clover (Trifolium incarnatum), and alsike clover (T. hybridum) are all very susceptible. White clover is generally considered to be less susceptible but not immune. Some other species of Sclerotinia may also occasionally infect clovers.

The disease is commonly referred to as a crown and stem rot, but it can attack all parts of the plant. Symptoms first appear in the fall as small, brown spots on the leaves and petioles. The heavily infected leaves turn grayish brown, wither, and become overrun with white mycelium, which spreads to the crowns and roots. By late winter or early spring the crowns and basal parts of the young stems show a brown, soft rot, which extends downward into the roots. Consequently part or all of the new growth of the infected plants wilts and dies. Stolons of Ladino clover may become soft and flaccid over their entire length or only small areas may be affected.

As the stems and petioles are killed, a mass of white mycelium grows over them. Some of the masses of mycelium then change into small, hard, black, cartilaginous bodies—the sclerotia. They are attached to the surface of (or imbedded in) the dead stems, crowns, and roots or in the soil near the roots. Some are as small as a clover seed. Some are larger than a pea seed.

When the affected plant parts decay, the sclerotia remain in the soil as a future source of infection. Sclerotia are the chief means by which the fungus survives from year to year. They can remain viable in soil for several years. In the fall, if conditions are right, the sclerotia germinate and produce one or more small, disk-shaped, pinkish-buff, mushroomlike fruiting bodies called apothecia, which are borne individually on slender stalks. The apothecia are one-sixteenth to one-fourth of an inch in diameter. They produce millions of spores, which spread to the leaves and petioles of nearby plants, causing infection and repeating the cycle.

The greatest reduction in stands occurs in late winter and early spring. The damage therefore is sometimes confused with winter killing. The disease usually occurs in patches throughout a field, but when conditions are very favorable the patches may become so numerous as to merge and cause extensive damage to a stand. In the South a few days of warm weather sometimes checks the disease, and the

plants recover.

Control is difficult. Clean cultivation, deep plowing to bury the sclerotia beyond their capacity to send up apothecia, and long rotations are helpful. Care should be taken not to distribute the sclerotia with clover seed. Grazing or clipping in late fall sometimes removes infected leaves and reduces the amount of foliage that may become infected and mat down on the crowns during the winter. Adapted varieties are more resistant than nonadapted strains. The most promising method of control appears to be the breeding of resistant strains.

Common root rot is a group of root diseases caused by species of *Fusarium* and several other soil fungi that produce similar symptoms and frequently attack plants simultaneously. The relative prevalence and importance of the fungi vary with the locality, kind of clover, age of plant, season of the year, soil type, and management practices. Mostly they are weak pathogens and cause damage after the plants have been weakened or injured. Most of them are widely distributed and cause damage wherever clovers are grown.

Scientists have learned a lot about the problem, but relatively little is understood concerning many of its phases. The field symptoms of common root rot of red clover are well known, for example, but attempts to reproduce them under controlled conditions often are unsuccessful. Research men have demonstrated that some isolates of the fungi can attack clover seedlings.

Species of *Fusarium* have been most frequently reported as causing root rot.

Symptoms of the disease are a localized or general rotting of any part of the root system. Taproot, secondary roots, and even the crown may be attacked. The color of the diseased areas ranges from light brown to black. The rotting may be limited to the cortical areas around the exterior of the root, the vascular core may be discolored, or the entire root may be affected. Secondary roots are constantly pruned away by the rots and new secondary roots are formed to compensate, but the replacement process is usually the slower, so that by the end of the second year most plants have left only a few short secondary roots. The lower part of the taproot often is destroyed completely. Such destruction causes wilting and a gradual dying of the plant.

Common root rot kills plants in all stages of development. Effects on stand are most conspicuous during the second year, but losses up to 45 percent during the first year are not uncommon. Stand losses occasionally occur in the spring when the plants are weak because of low food reserves or winter injury. Diseased stands frequently produce a fair first crop of hay but fail to recover and to produce a second crop. Most clovers and sweetclovers are susceptible to root rot.

Besides Fusarium, Rhizoctonia, Phoma, and other organisms may be associated with root rots. Plenodomus meliloti and Cylindrocarpon ehrenbergi are of primary importance following the winter dormancy period on sweetclover in Alberta, Canada.

Control is difficult, but any practice

that improves the general vigor of the plant is helpful. Proper liming, fertilization, and crop rotation are important. Only adapted varieties should be grown. No varieties available in 1953 had high resistance when conditions favor the disease. Plant breeders have under way a project to develop resistant strains of red clover.

Phytophthora root rot, caused by *Phytophthora cactorum*, is a widespread disease of sweetclover in North America, notably Ohio, Indiana, Illinois, and Missouri. It occurs in Alberta and Ontario. The fungus attacks individual plants or small groups of plants in fields or along roadsides. It is most abundant in low, wet parts of fields, where in seasons of heavy spring rainfall and cool temperatures it may kill most of the plants.

Its presence is first noted in the spring, when infected plants wilt, die, or are generally unthrifty. When their roots are examined, the upper portions usually are found to be rotted. The decay generally is limited to the upper 3 or 4 inches but may extend as much as 8 inches below the crown. The decayed places usually are soft and watery. The color changes but little at first. Later they may become discolored and shrunken.

Crop rotation and the use of well-drained fields are helpful control measures. It should also be possible to develop resistant varieties because resistant plants are known to exist.

Seedling blights, caused by Pythium, Rhizoctonia, and other fungi, are the most destructive seedling diseases. They occur wherever clovers grown. Sometimes they seriously reduce the stands. Three types of injury occur. Preemergence killing starts shortly after the seed is sown and develops rapidly, so that the seedlings are destroyed before they emerge from the soil. In postemergence dampingoff, infection commonly occurs before emergence, although the rate of disease development is slower and the seedlings emerge only to be killed soon thereafter. Root and hypocotyl rotting

causes varying degrees of stunting, in which plants survive the early seedling stages, after which some recover and some die.

Seedling blights are caused by a complex of fungi, including several species of *Pythium*, *Rhizoctonia*, *Fusarium*, *Gliocladium*, *Phoma*, and others. One of the most virulent is *Pythium debaryanum*. It would seem that seed treatment might help control this group of diseases, but the results of field tests have given limited encouragement for this method of control.

The STEM diseases attack the supporting and conducting systems of the plant. Often they cause serious losses. Usual symptoms are stem discoloration, withering and dying of attached leaves and petioles, and general wilting and stunting of the plant. Frequently stems break off or crack open at the site of infection. Several of the major diseases of clover are included in this group.

Northern anthracnose, caused by Kabatiella caulivora, is a major disease of red clover in the cooler areas of North America, Europe, and Asia. It develops best at 68° to 77° F. and is checked by continuous hot dry weather. In the United States it is important only in the northern clover regions but there it frequently causes damage occasionally exceeding 50 percent of the crop in some fields. Losses as high as 50 to 60 percent have been reported in Germany. Complete crop failures have been observed in the Netherlands. Seed production and hay yield and quality are greatly reduced in badly infected fields.

The disease is serious only on red clover. It may occur on alsike, white, crimson, and Persian (*Trifolium resu-pinatum*) clovers and possibly others. It has never been found on alfalfa, but has been reported on black medic and on sainfoin in the Netherlands. The species of fungus inciting the disease consists of a large number of physiologic races, which differ in their capacity to infect different species of clover

and different strains of a particular species. No red clover strain yet developed is immune to the disease, but wide differences in resistance exist among European and American strains. Varieties developed in the southern part of the United States are more susceptible than those developed in the northern clover areas.

Symptoms are confined mostly to the petioles and stems. Infection also occurs on the petiolules-small stalks connecting the leaflets to the petiole and occasionally on the leaflets themselves. The first symptoms noticed in the field are usually dark-brown or black spots on the petioles. The spots soon cut off translocation to the parts above them—the upper part of the petiole and the leaf—causing them to wilt, turn grayish brown, and die. The petiole bends downward at the site of the lesion to form the familiar "shepherd's crook." Stem lesions are most characteristic. They develop first as small, dark spots, which soon lengthen to form lesions with dark margins and light-colored centers. As the stem grows, a crack often appears in the center of the lesion. Stems finally may be girdled and killed. Plants in a badly infected field look as if they were scorched with fire, because of the abundance of blackened and broken stems, withered petioles, and brown, dead leaves. The name scorch has been aptly used in Britain to denote the disease.

Southern anthracnose, caused by Colletotrichum trifolii, is a major disease of red clover in the southern clover belt of the United States. It has been recorded as far north as southern Canada, but is primarily a high-temperature disease that flourishes at about 82° F. It is of little economic importance in the northern clover areas. It is confined mostly to North America, although it has been reported on alfalfa in South Africa and in Europe. It occurs occasionally on crimson clover, sub clover (Trifolium subterraneum), bur-clover (Medicago hispida), and white sweetclover (Melilotus alba). It has not been observed on white clover. Alsike clover is practically immune.

Southern anthracnose has been regarded as the most destructive disease of red clover in the Southern States. It reduces yields of hay and seed and can destroy stands of clover. A resistant variety, Kenland, is available. Most European and American strains developed in regions where the disease does not occur are susceptible; hence it is important to grow only locally adapted strains or strains known to be resistant.

Symptoms resemble those of northern anthracnose; in fact, a positive identification in the field is frequently difficult and sometimes impossible. Dark tufts of setae in the older lesions indicate that the disease is southern anthracnose. But there are other distinguishing features. Southern anthracnose commonly attacks the upper part of the taproot; that has not been observed for northern anthracnose. Southern anthracnose usually produces more spotting of the leaves, but that is not an infallible characteristic because of the frequent presence of similar leaf spots incited by other pathogens. Like northern anthracnose, it may occur on plants at any stage of development. It most commonly develops on the young, succulent parts of stems and petioles but is not limited to them.

The disease occurs on the leaves as dark-brown spots of irregular shape, which vary from pin-point lesions to a general infection over most of the surface. Petioles are very susceptible. They become dark brown, and the attached leaflets droop. First symptoms on the stems and petioles are small, water-soaked spots, which usually lengthen to form long, depressed, dark-brown or black lesions, many of which develop gray or light-brown centers. Lesions near the base of a stem often cause death and browning of the entire stem.

The most destructive effect of southern anthracnose is on the taproot and crown. Dark lesions develop on the upper part of the taproot, gradually girdle it, and cause the plant to wilt and die. This crown rot is closely associated with taproot decay and may result from spread of the fungus upward from the roots or downward from the stems and petioles. Diseased crowns become brittle so that the stems are readily broken off at the soil level. Crown and root rot caused by southern anthracnose kills some plants and weakens others so that they cannot survive long drought, adverse winter conditions, and attacks of other diseases.

Black stem, caused by *Phoma*, *Mycosphaerella*, and *Ascochyta*, is a major disease of clovers. It is widely distributed and may cause extensive damage during cool, wet weather in the fall, late winter, and spring. It causes the familiar stem blackening and repeated defoliation, which weakens and sometimes destroys stands. The disease was so severe in Kentucky in 1933 on some of the unadapted red clovers that plots that had had perfect stands the previous December were bare by April.

Among the fungi that cause black stem of clovers are Ascochyta imperfecta, which occurs mostly on alfalfa but sometimes on clovers; Phoma trifolii, the organism most frequently attacking red clover; and Mycosphaerella lethalis, the cause of black stem of sweetclover. Little is known about the host range of these pathogens except that each of them can infect alfalfa, red clover, and sweetclover and each is primarily the cause of the disease on its own crop. During midsummer and fall, another pathogen, Cercospora, also causes black stem.

The most conspicuous symptom is stem blackening, which may involve all or any part of the stem. Blackening increases when clover is not cut at the proper time or when the crop is left for seed. Frequently young shoots or petioles are girdled and killed. This, as well as leaf infection, may result in severe defoliation.

On red clover the disease produces

small, dark-brown or black spots, which increase slowly in size and eventually kill the affected parts. Infection occurs the first summer on spring-sown clover but becomes more destructive the following late winter and spring. On unadapted clovers, new leaves may be killed as rapidly as they are formed. On sweetclover the disease appears most commonly in the spring of the second year. The spots at first are dark. As they enlarge they change to light brown. Leaf spotting is increased by frost injury, which seems to provide an avenue of entrance for the fungus. Heavy stands may be greatly injured. The disease is more severe on plants that have been clipped or grazed.

Crop rotation and burning of dead leaves and stems before new growth develops in the spring are helpful control measures. Breeding for resistance has been started.

Stem canker, or gooseneck, is caused by *Ascochyta caulicola*. It was first reported in Germany in 1903 as a new disease of sweetclover. It is now known to occur in most areas of the world where sweetclover is grown. It has not been observed on other legumes.

It produces silvery-white cankers on the stems, petioles, and occasionally the midribs of the leaves. The cankers vary in size. They are stippled with numerous tiny black dots and have brown margins. On the lower parts of the stems the cankers may be so large and numerous as to girdle the stems. On the upper parts they are less abundant, smaller, and more isolated. Heavily infected stems often appear swollen, are retarded in development, and have fewer and smaller leaves. They also tend to twist and bend at the top.

LEAF DISEASES usually do not kill plants, but they interfere with the normal functions of the leaf. Sometimes they cause defoliation, which reduces yield, quality, and palatability of the forage. If the defoliation is extensive and continuous, the plants lose vigor, are less able to survive unfavorable

conditions, and are more readily attacked by pathogens that cause root rots.

Pseudopeziza leaf spot, caused by Pseudopeziza trifolii, is widespread in the cooler, humid clover regions of the United States and Europe. It has been reported also from Canada and Russia. It usually is of minor importance, but occasionally severe local outbreaks cause extensive defoliation. Serious outbreaks have occurred in northern Indiana, Ohio, and the Northeastern States. It has been called the most serious leaf disease of red clover in New York.

It resembles the pseudopeziza leaf spot of alfalfa, but it does not attack alfalfa and the disease of alfalfa does not attack the clovers. It occurs on red, alsike, white, crimson, zigzag (*Trifolium medium*), and strawberry (*T. fragiferum*) clovers and several others. A similar disease of sweetclover is caused by *P. meliloti*.

Dark spots that may be olive to reddish brown, purple, or black develop on either leaf surface. The spots are tiny, angular, or round and commonly have dendritic margins. A minute, amber, jellylike globule occurs in the center of the older spots. The globules, or fruiting bodies, more frequently are found on the lower side of the leaf but occasionally occur on both sides. They are most abundant in wet weather. Later they dry up, shrink, become almost black, and are not readily detected. Positive diagnosis of the disease in its early stages in the field is difficult because at first the minute pin-point lesions are not markedly different from those of other leaf spots. The disease is almost entirely limited to the leaves, but has been reported to produce small, long, dark streaks on the petioles.

Stemphylium leaf spot, or target spot, is caused by *Stemphylium sarciniforme*. It is common on red clover in the United States and Europe. It is not considered a major disease of red clover, but its importance may have been underestimated. It can cause serious defoliation and losses of 15 to 40 percent of the

crop in individual fields. It is known to occur in nature only on red clover. L. J. Krakover, working at the Michigan Agricultural Experiment Station at East Lansing, inoculated sweetclover, alsike, white, and crimson clovers, as well as alfalfa, vetch, and several other legumes, but he was not able to infect them. James G. Horsfall, at the New York Agricultural Experiment Station at Ithaca, however, reported infection from artificial inoculations on alsike and white clovers, sweetclover, and alfalfa. That difference in ability of the isolates to cause disease suggests that the fungus may have more than one race.

Symptoms are limited almost exclusively to the leaflets. Minute, lightbrown spots, similar to the early symptoms of some of the other leaf spot diseases, appear first. Fully developed spots are mostly oval or round and about one-fifth inch across. They may be larger when only a few occur on a leaf. The most characteristic symptom is the occurrence of concentric rings within the lesion, suggesting the name target spot. The center of a typical spot is dark and distinct. It is surrounded by alternately light and dark rings. The darker rings are sepia to dark brown. The lighter ones are ocher to light brown. The darker rings near the center of the lesion are narrow and ridged. The color contrast between the two outermost rings is very sharp. Spots are most abundant near the margin of a leaf but may occur anywhere. Frequently they coalesce, killing large areas of the leaf and causing defoliation. Symptoms on the stems and petioles are uncommon but when they do occur they appear as dark-brown to black linear streaks.

A related fungus, S. botryosum, attacks alfalfa and sometimes occurs on red clover.

Blackpatch, caused by an unidentified fungus, was first recognized in Kentucky as a disease of red and white clovers. It has also been reported from Wisconsin, West Virginia, and Georgia. It has generally been considered

of little economic importance but occasionally causes losses in local areas. In heavily infected fields of red clover the seed yield may be reduced at least

50 percent.

In addition to red and white clovers, the disease has been found on soybean (Glycine max), cowpea (Vigna sinensis), kudzu (Pueraria thunbergiana), and blue lupine (Lupinus angustifolius). It has not yet been reported as occurring in nature on alsike clover, crimson clover, alfalfa, and the sweetclovers, but all of those crops have been infected by artificial inoculation.

Blackpatch attacks the leaves, stems, flowers, and seeds. Under normal conditions it occurs in patches. Otherwise it appears only on scattered plants. Leaf lesions are similar in size and color to those caused by Stemphylium sarciniforme. They vary from brown to grayish black and usually have concentric rings. Large areas of a leaf may be affected. Sometimes all the lower leaves are killed. Greatest damage results from the girdling of the stems beneath the flower head or from direct infection of the flowers before the seeds are fully developed. The fungus is seed-transmitted. It also causes seedling blight. Examination of diseased plant parts with a hand lens usually reveals the presence of coarse, dark, aerial mycelium, a characteristic that helps in diagnosing the disease.

Treating the seed with a fungicide should aid in preventing initial infection. In hayfields, losses can be reduced by early harvesting. Crop rotation and sanitation should also be helpful.

Curvularia leaf spot is caused by Curvularia trifolii. It sometimes causes considerable wilting and premature dying of leaves of Ladino clover in the eastern United States. It was first discovered in 1919, when it caused minor damage to white clover near Washington, D. C. Since 1940 it has occurred more frequently, presumably because of the widespread use of Ladino clover along the Atlantic seaboard. Up to 25 percent of the leaves may be attacked and damaged. Ladino

clover may be more susceptible than common white clover. It is possible to infect other species of clover in the laboratory, but the disease has not been found attacking them in the field. Infected leaves usually have a large yellowed area, which soon turns watery gray and translucent, then light brown. A yellowish band usually outlines the advancing edge of the infected part of a leaf. Diseased areas that originate at a leaf tip sometimes become V-shaped. Infected leaves wilt, then shrivel and die. Sometimes the dead V-shaped part of a leaf curls. The fungus can invade the entire leaflet and grow down the petiole, causing complete wilting of the leaf. Apparently it does not attack stolons. The disease develops most rapidly during warm, wet weather. A temperature of 75°-80° F. is most favorable.

Cercospora leaf and stem spot, also called summer black stem, is widely distributed in the United States and Europe. It is commonly found on most of the true clovers, including red, alsike, white, crimson, zigzag, hop, (Trifolium agrarium), and many others. A similar disease occurs on alfalfa, sweetclover, black medic, and related species. Cercospora zebrina is the most important pathogen on all of those hosts except sweetclover. C. davisii is the most important (or the only) species infecting sweetclover. Damage varies from year to year, depending on weather conditions, but the disease is always present and frequently causes excessive premature defoliation.

Symptoms vary somewhat on the different host plants. Leaves, stems, petioles, petioles, and seeds may be attacked. Leaf lesions are usually angular and more or less confined by the veins. The size and shape of the lesions vary from rather small, linear spots on red clover to large, almost circular ones on sweetclover. Apparently atmospheric conditions and the kind of tissue influence the size and shape of the lesions. Color of the spots also varies considerably. On the true clovers the general tone is reddish or

smoky brown. On sweetclover it is ashy gray. When conditions favor sporulation of the pathogens, a silverygray down develops on mature lesions. Lesions on the stems and petioles are somewhat sunken but with colors like those of the leaf spots. Stem infections are serious as they cause the distal parts to wilt and die. Seeds may also be infected. The disease may be distributed on the seed.

Infection may occur at any time during the growing season on plants of any age, but the disease is usually most abundant in late summer and autumn. On sweetclover the disease is most conspicuous on second-year plants after they have started to bloom and is more severe on plants that have been cut or grazed.

Little is known concerning the control of this leaf and stem spot. Removing old crop residues and crop rotation help reduce damage. Infected seed should be treated with a fungicide to reduce seedling infection. The development of resistant varieties of

legumes seems to hold out the most

hope for controlling it.

Pseudoplea leaf spot, or pepper spot, is due to *Pseudoplea trifolii*, a fungus that attacks the true clovers and less commonly alfalfa. It occurs throughout the United States, Canada, Europe, and Asia. The disease has not been reported on sweetclover. It is important on Ladino and white clover in Northeastern and Southern States, where severe infection frequently causes premature yellowing and defoliation of lower leaves. It occurs throughout the growing season but develops most abundantly in cool, wet weather.

Tiny, sunken black flecks develop on both surfaces of leaves and on petioles. The flecks rarely reach a diameter of more than a few millimeters but frequently are very numerous—hence the name pepper spot. Later they turn gray with a dark, reddish-brown margin. Heavily infected leaves and petioles become yellow, wither, turn brown, and collapse as a dead mass. Flower stalks and floral parts may also become infected and be killed.

Infection occurs from spores that develop on dead, overwintered leaves and petioles. Spots can usually be found on the first new leaves that emerge in the spring.

No practical control measures are known, but plants differ in susceptibility, and breeding for resistance is

possible.

Bacterial leaf spot, caused by *Pseudomonas syringae*, does not usually cause serious damage, although it is widespread in the United States and has been reported in Italy and England. Wet weather favors its rapid spread and development. Hot, dry weather checks it.

The disease may appear at any time during the growing season. It is most conspicuous on the leaflets. It also affects the stems, petioles, petiolules, stipules, and flower pedicels. First symptoms are tiny, translucent dots on the lower leaf surface. The spots enlarge and fill the angles between the veins. They are tiny and black except for the margins, which retain a water-soaked appearance. In wet weather a milky-white bacterial exudate may develop as a thin film or as droplets. On drying, the exudate becomes a thin, incrusting film, which glistens in the light. Tissues surrounding the spots are yellowish green. Infection may be so abundant that whole sections of a leaflet are killed. Mature leaves are often perforated and frayed because of the drying and shattering of parts of the diseased tissues. Lesions on the petioles and stems are dark, elongated, and slightly sunken.

Several clovers, including red, alsike, white, crimson, zigzag, and Berseem (*Trifolium alexandrinum*), are known hosts of this pathogen. Isolates from different areas differ in pathogenicity.

Several other bacterial diseases have been found on clovers, but none of them is important in America.

Sooty blotch, caused by Cymadothea

trifolii, is one of the most conspicuous and easily identified leaf spots of clover. It is prevalent throughout North America, particularly in the southern part of the United States, and in Europe. It is most common in alsike, red, and white clovers. Frequently it reaches epidemic proportions on crimson clover. It also has been reported as occurring on some 24 other true clovers.

In the Southern States the disease appears in the spring. In Northern States it is more prevalent in late summer and fall. The earliest symptom is the development of minute, olive-green dots mostly on the lower leaf surface. The dots enlarge and become thicker and darker until they acquire the appearance of velvety black, angular, elevated patches or warts. In the fall the warts are replaced by other black areas, which have a shiny surface. Chlorotic and later necrotic spots appear on the upper surface of the leaf immediately above the warts. When spots are abundant the entire leaf may turn brown, die, and fall off. Sooty blotch is of considerable economic importance on crimson clover, causing reduction in seed yield.

Powdery mildew, caused by Erysiphe polygoni, is a common and widespread disease of clovers. It probably occurs wherever they are grown. It can cause reductions in the yield and quality of hay. Ordinarily it is of little consequence on the first hay crop but is more abundant on the second. It can attack plants at any stage of maturity but develops best during the cool nights and warm days of the latter half of summer and fall. Long spells of dry weather favor its development.

The pathogen has been recorded on some 359 species belonging to 154 genera. It consists of a number of physiologic races, which differ in their ability to attack different genera and species of hosts and different varieties of a species. European varieties of red clover have been reported generally to be more resistant than American

varieties, but American strains are now available that are highly resistant. Wisconsin Mildew Resistant is one of them. Most varieties contain a few resistant plants.

At first barely perceptible patches of fine, white, cobwebby mycelium develop on the upper surface of the leaves. The patches enlarge and merge, the fungus sporulates, and the leaf surface appears as if it had been dusted with white flour. Symptoms may also occur on the lower surface of leaves. Severe attacks can make whole fields appear white.

Rusts of clovers are widely distributed in the humid and subhumid areas of the world. Damage is difficult to assess because heavy infection usually does not occur until late in the summer. Occasionally heavy infection occurs, and severe loss results when a grower attempts to produce two seed crops in a season and leaves the accumulated rust-infected old growth to infect the new growth.

Three common varieties of rust attack clovers. They cannot be distinguished on the basis of symptoms but can be differentiated by differences in their capacity to infect the various clovers. For example, the variety of rust on alsike clover (Uromyces trifolii hybridi) infects only alsike, while the rust on red clover (U. trifolii fallens) infects red, zigzag, crimson, Berseem, and several other clovers. The rust on white clover (U. trifolii trifolii-repentis) does not infect red or zigzag clovers but does infect crimson and Berseem, besides white clover. These rust fungi differ from those causing the cereal rusts in that they can complete their entire life cycle on a clover species and do not require an alternate host.

The most conspicuous symptom of clover rust is the uredial, or brown rust, stage, in which round or irregular, pale-brown pustules, surrounded by the torn epidermis, appear on the lower surface of leaves and on the petioles and the stems. Sometimes in

winter in the South and early spring in the North, small swollen whitish-toyellow clusters of tiny cuplike structures occur on the stems, petioles, and large veins of the leaves. These are called the aecia and may cause distortion of the affected leaves and petioles. Rust fungi in the telial, or black spore, stage overwinter on the debris of diseased plants.

Fungicides such as sulfur can be used to control the clover rusts, but rarely is it practical to use them. Resistant plants exist in present varieties and resistant strains can be developed if the importance of the disease warrants it.

All common clovers are susceptible to several viruses, some of which are widely distributed. Most of the legume viruses have a wide range of hosts—so that clover viruses infect not only clovers but also other legumes, and viruses of other legumes attack clovers. Some viruses from nonlegume hosts such as tobacco, gladiolus, potato, and some weeds can readily infect clovers. That means that many of the virus diseases spread from one crop to another. The extent of the spread and amount of infection usually depend on the kinds and numbers of insects present. Aphids are probably the most important carriers.

Symptoms vary with the virus and host. Most of the clover viruses are systemic—that is, they are present in all parts of the plant. The most conspicuous symptoms are usually found in the leaves. They include vein chlorosis, mild to severe mottling, chlorotic patches between the veins, and other abnormal combinations. Sometimes the leaves are curled, puckered, or ruffled. Some viruses cause a reduction in vigor as indicated by a general stunting of the plant. Others have no apparent effect on vigor. Viruses that have little effect on one clover may kill another. Symptoms of most clover virus diseases are conspicuous during the cooler periods of the growing season and sometimes disappear temporarily or are masked during hot weather. Weakening caused by virus disease may predispose plants to attack by other pathogens (especially those that cause root rots) or prevent them from surviving severe winters or prolonged droughts.

The virus diseases reported to occur on the more important clovers are:

Red clover: Red clover vein-mosaic, common pea mosaic, yellow bean mosaic, potato yellow dwarf, American pea streak, New Zealand pea streak, Wisconsin pea streak, pea mottle, pea wilt, alfalfa mosaic, sub clover mosaic, ring spot, broadbean common mosaic, broadbean mild mosaic, and cucumber mosaic.

Alsike clover: Alsike clover mosaic, red clover vein-mosaic, sub clover mosaic, common pea mosaic, pea mottle, pea wilt, and New Zealand pea streak.

White clover: Alfalfa mosaic, red clover vein-mosaic, yellow bean mosaic, pea mottle, pea wilt, American pea streak, New Zealand pea streak, and broadbean mild mosaic.

Crimson clover: Alfalfa mosaic, red clover vein-mosaic, sub clover mosaic, alsike clover mosaic, common pea mosaic, yellow bean mosaic, pea mottle, pea wilt, American pea streak, potato yellow dwarf, and broadbean common mosaic.

Sub clover: Sub clover mosaic and yellow bean mosaic.

Sweetclovers: Alfalfa mosaic, alsike clover mosaic, red clover vein-mosaic, sub clover mosaic, common pea mosaic yellow bean mosaic, pea mottle, pea wilt, American pea streak, tobacco streak, tobacco ring spot, and broadbean mild mosaic.

Little has been done to control the virus diseases of clovers. Some of the newer insecticides kill the insect vectors. When possible, clover should not be grown close to other legumes, particularly peas or beans. The ultimate solution is to develop varieties of clovers resistant to the most prevalent and injurious virus diseases. That remains to be done.

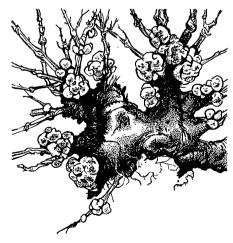
SEVERAL PATHOGENS can attack the floral parts of the clovers under some

conditions. They are of importance only when they interfere with seed production.

Anther mold (Botrytis anthophila) was first reported from Russia in 1914 and has since been found to be widely distributed in Europe. In the United States it has been found to a limited extent in Oregon. It has no apparent effect on the plants until flowering time, when it destroys the normally yellow pollen grains, replacing them with the gray spores of the fungus. If abundant it can reduce seed setting.

Earle W. Hanson, a native of Minnesota, joined the Department of Agriculture in 1937. From 1937 to 1946 he was employed by the division of cereal crops and diseases of the Bureau of Plant Industry, Soils, and Agricultural Engineering doing research at the Minnesota Agricultural Experiment Station on the diseases of hard red spring wheats and the development of disease-resistant varieties of wheat. Since 1946 he has been employed jointly by the division of forage crops and diseases of the Bureau and the University of Wisconsin.

KERMIT W. KREITLOW is also a member of the division of forage crops and diseases and is stationed at Beltsville, Md. He is a graduate of the University of Minnesota and Louisiana State University. Dr. Kreitlow has been engaged in work on forage crop diseases since 1941.



Crown wart of alfalfa.

Sources of Healthier Alfalfa

Fred R. Jones, Oliver F. Smith

Alfalfa as a forage crop in the United States is recognized as consisting of strains of Medicago sativa and of hybrids of that species with Medicago falcata. M. sativa occupies the southern and central alfalfa regions. The apparent hybrids appear to use the superior hardiness of the M. falcata parent to give them longevity in the severe climate of the northern part of the range of the crop. Thus two species, which vary greatly within themselves, produce in their combined resources a crop that in the hands of progressive agriculturists has spread across a wide range of climate.

As often happens in such wide and intensive culture, serious diseases have appeared. Many have reduced the quantity and quality of alfalfa forage. To overcome the loss, breeding for resistance has been undertaken. Already those efforts have demonstrated that the qualities of two variable species can be utilized to develop resistance to many of the diseases.

The evaluation of the resistant characters is one of the major tasks that face pathologists and breeders. Therefore in this discussion of diseases of alfalfa, we emphasize sources of resistance.

Of the nonparasitic diseases, winter injury is often the cause of weak growth in the spring and the subsequent unthrifty condition of many plants. Besides, injured tissues often become the avenues of entrance for